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EXPERIENCE IN MANAGING PRIMARY GRAFT DYSFUNCTION AFTER HEART TRANSPLANTATION

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Heart transplantation (HT) is a cardiac surgical procedure involving the replacement of a recipient's pathologically impaired heart with a functionally adequate donor organ. As with any major surgical intervention, HT comes with possible complications, one of which is primary graft dysfunction (PGD). This report presents our initial experience in the diagnosis and management of a patient who developed PGD, necessitating the use of mechanical circulatory support.

Keywords: heart transplantation, primary graft dysfunction, mechanical circulatory support, ECMO, acute kidney injury, renal replacement therapy.

INTRODUCTION

Heart transplantation (HT) is a surgical procedure in which a pathologically altered heart is replaced with a viable donor heart. It is primarily indicated for patients with end-stage heart failure – classified as stage IIB–III according to the Vasilenko–Strazhesko system – and who experience significant limitations in physical activity (NYHA functional class III–IV). Candidates typically exhibit poor response to pharmacological therapy or mechanical circulatory support (MCS), and are not suitable for other surgical interventions, yet possess the potential for clinical remission following transplantation [1]. HT remains the gold standard in the management of end-stage chronic heart failure (HF).

Like any major surgical procedure, HT is associated with the risk of complications, among which primary graft dysfunction (PGD) is one of the most serious. According to various studies, PGD incidence ranges from 2.3% to 32.4% [2–6]. PGD is defined as mono- or biventricular dysfunction of the allograft occurring within the first 24 hours post-transplantation. This condition results in hypotension due to inadequate cardiac output that fails to meet the recipient's circulatory needs [7].

Despite advances in perioperative management, particularly regarding immunosuppressive therapy, the risk of early graft dysfunction remains significant, with a reported 30-day mortality rate of 5–10% [8]. Between 2017 and 2023, a total of 13 heart transplants were performed at Chelyabinsk Regional Clinical Hospital. PGD occurred in only one case. This report presents our first clinical experience with the diagnosis and management of PGD requiring MCS.

CLINICAL CASE

Patient B., a 53-year-old male, was placed on the heart transplant waiting list in April 2023 with a diagnosis of coronary artery disease and ischemic cardiomyopathy, classified as HF stage IIa, functional class III.

In June 2023, the patient was admitted to the cardiac surgery department of Chelyabinsk Regional Clinical Hospital for a heart transplant.

During the preoperative period, he underwent a standard pre-transplant evaluation. On the day of surgery, he was transported to the operating room. Upon arrival and transfer to the operating table, the patient remained stable. An initial anesthesiology assessment revealed a sinus rhythm with a heart rate of 70 beats per minute. Oxygen saturation was 97% on room air (FiO_2 21%). Preoxygenation was initiated via face mask.

The left radial artery was catheterized to enable invasive blood pressure monitoring and facilitate blood sampling for laboratory analysis. Standard induction of anesthesia was carried out, and the patient was transitioned to mechanical ventilation using lung-protective parameters.

Initial central hemodynamic assessment was performed using a Swan–Ganz catheter. At the time of measurement, the patient was receiving inotropic support with dobutamine at 5 μg/kg/min and vasopressor support with norepinephrine at 0.03 μg/kg/min. The following hemodynamic parameters were recorded: central venous pressure (CVP) = 14/9 mmHg; right ventricular systolic/diastolic pressure ($RVP_{sys/dia}$) = 25/10 (mean 17) mmHg; pulmonary artery systolic/diastolic pressure ($PAP_{sys/dia}$) = 27/18 (mean 19) mmHg; pulmonary capillary wedge pressure ($PCWP_{sys/dia}$) = 16/14 (mean 12) mmHg; cardiac output (CO) = 2.9 L/min; cardiac index (CI) = 1.5 L/min/m^2.

Intraoperatively, immunosuppression was initiated in accordance with the protocol developed at Shumakov National Medical Research Center of Transplantology

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and Artificial Organs, Moscow, Russian Federation. The protocol included:

1. *Administration of basiliximab 2 hours prior to HT.*
2. *Administration of methylprednisolone 1000 mg before aortic unclamping.*

Hemodynamic parameters remained stable throughout the pre-perfusion period. Infusion therapy followed a restrictive strategy, with a total infusion volume of 300 mL administered prior to initiation of cardiopulmonary bypass (CPB).

After administration of heparin and achievement of the target activated clotting time (ACT), CPB was initiated as planned. The procedure was performed under mild hypothermia (33–34 °C), with a perfusion rate ranging from 4.6 to 6.3 L/min (100–120% of the target flow). Mean arterial perfusion pressure was maintained at 50–70 mmHg.

At the time of aortic unclamping, the patient had been rewarmed to 36.0 °C. Methylprednisolone was administered at a dose of 1 g. After heart deaeration procedure, the aortic cross-clamp was removed. Total cold ischemia time of the donor heart was 2 hours and 37 minutes.

During reperfusion of the transplant, spontaneous cardiac activity resumed; however, it was accompanied by signs of atrioventricular block, with a heart rate of 25–30 beats per minute prior to the initiation of inotropic support with dobutamine at 10 μg/kg/min. Due to the inadequate heart rate and rhythm, dual-chamber epicardial pacing was initiated at a rate of 90–100 beats per minute using epicardial electrodes.

Following 30 minutes of reperfusion – during which the patient's blood pressure was maintained at 90/60 mmHg and CVP at 16/10 mmHg – CPB was discontinued. At that point, cardiotonic support included epinephrine at 0.03 μg/kg/min, norepinephrine at 0.1 μg/kg/min, and dobutamine at 5 μg/kg/min, yielding a vasoactive-inotropic score (VIS) of 18. Fractional administration of protamine sulfate was initiated.

Subsequently, a gradual decrease in arterial pressure to 80/50 mmHg was observed, despite an escalation in inotropic and vasopressor support: epinephrine increased to 0.05 μg/kg/min, norepinephrine to 0.3 μg/kg/min, and dobutamine to 20 μg/kg/min (VIS = 55). At the same time, 100 mg of protamine sulfate was administered over 10 minutes. To rule out peripheral vasospasm as a contributing factor, the left femoral artery was punctured and catheterized. No significant difference in invasive blood pressure was observed when measured in the supine position. At this stage, graft dysfunction was suspected.

Due to persistent hemodynamic instability, an increase in VIS to 55, and worsening signs of hypoperfusion, evidenced by progressive hyperlactatemia (increasing from 5.2 to 7.9 mmol/L), veno-arterial extracorporeal membrane oxygenation (VA-ECMO) was initiated. Prior to ECMO initiation, central hemodynamic parameters were reassessed, which further confirmed the diagnosis of graft dysfunction: $CVP_{sys/dia}$ = 16/14 (mean 12) mmHg; $RVP_{sys/dia}$ = 42/24 (mean 28) mmHg; $PAP_{sys/dia}$ = 45/40 (mean 30) mmHg; $PCWP_{sys/dia}$ = 30/28 (mean 20) mmHg; transpulmonary gradient (TPG) = 10 mmHg, CO = 3.8 L/min; CI = 2.1 L/min/m^2.

After achieving an ACT of 160 seconds, VA-ECMO was initiated via central cannulation. The targeted volumetric perfusion rate was 2.4 L/min/m^2 (approximately 5 L/min total flow). ECMO support was provided using a Maquet system equipped with a Maquet PLS ECMO circuit (Maquet AG, Germany).

To minimize postoperative blood loss and reduce the risk of infectious complications, the surgical wound was closed using polypastic sutures to secure the sternal fragments.

In the intensive care unit (ICU), the patient continued VA-ECMO at a flow rate of 2.4–2.6 L/min/m^2. On postoperative day 1, levosimendan was administered at a dose of 2 μg/kg/min. After initiation of ECMO, there was a marked reduction in cardiotonic support requirements, with the Levosimendan Vasoactive-Inotropic Score (LVIS) decreasing to 24–23 points. Perfusion pressure was maintained at 90/70 mmHg, despite minimal native cardiac output. Sinus rhythm was restored within the first 24 hours. Mechanical ventilation was provided using a lung-protective volume-controlled mode.

On postoperative day 2, transthoracic echocardiography was performed. The findings showed no significant changes compared to the initial assessment of the donor heart. Comparative echocardiographic parameters are summarized in Table.

By day 3, a progressive decline in urine output was observed, despite pharmacological stimulation.

By the beginning of postoperative day 4, cardiotonic support had been significantly tapered, consisting only of a dobutamine infusion at 3 μg/kg/min. MCS was also reduced to 1.0 L/min/m^2. Despite these improvements, the trend of reduced urine output persisted.

Given the stabilization of hemodynamic parameters and the absence of arrhythmias, a decision was made to initiate weaning from ECMO on day 4 of ECMO therapy. After volume loading and an increase in inotropic therapy to achieve a VIS of 25, ECMO was successfully discontinued. Additional volume loading allowed for a subsequent reduction in inotropic support, lowering the VIS to 10.

Following ECMO weaning, transesophageal echocardiography was performed. Findings included thickening of the left ventricular walls and interventricular septum, with a reduction in left ventricular volume: left ventricular end-diastolic volume (LVEDV) = 50.0 mL; left ventricular end-systolic volume (LVESV) = 32.0 mL; interventricular septal thickness: 1.6 cm; posterior wall thickness: 1.5 cm; ejection fraction (EF) = 51% (Simpson's method); tricuspid annular plane systolic excursion (TAPSE): 1.6–1.8 cm. Blood pressure was

maintained at ≥95/65 mmHg with moderately increased catecholamine support compared to intraoperative levels (VIS = 14).

By postoperative day 5, the patient developed acute kidney injury. Glomerular filtration rate (GFR) declined to 18 mL/min/1.73 m^2, accompanied by a reduction in urine output to 0.5–0.7 mL/kg/hour, despite increased diuretic stimulation with furosemide at 20 mg/hour. Pulmonary oxygenation deteriorated significantly, with a drop in the P/F ratio to 127. In response, a session of prolonged veno-venous hemofiltration was initiated using the PrismaFlex system (Gambro Lundia AB, Sweden).

A telemedicine consultation was held with Professor V.N. Poptsov, MD, from Shumakov National Medical Research Center of Transplantology and Artificial Organs, Moscow, Russian Federation, Moscow, during which indications for the resumption of MCS were established.

However, given the lack of a rising trend in inotropic requirements, the predominance of respiratory distress and renal failure in the clinical picture, and the extremely high risk of infectious complications, it was decided to discontinue MCS and to continue with prolonged veno-venous hemodiafiltration.

On postoperative day 6, central hemodynamic monitoring revealed an increase in both cardiac output and cardiac index, achieved with relatively low levels of inotropic support (VIS = 15).

By day 7, the session of high volume venovenous hemodiafiltration (HV-CVVHDF) was discontinued. It lasted for 52 hours, during which hemofiltration rate was maintained at 30 mL/kg/h. A gradual return of spontaneous diuresis was observed, along with a continued decrease in inotropic requirements (VIS = 11). However, respiratory failure persisted, with a P/F ratio of 170.

On day 8, a tracheostomy was performed. By that time, inotropic support had been reduced to a minimal level (VIS = 2), and oxygenation had significantly improved, with the P/F ratio rising to 300.

Three days later, a downward trend in serum creatinine and urea levels was observed, eliminating the need for further renal replacement therapy.

Over the next eight days, efforts were focused on weaning the patient from mechanical ventilation using high-flow oxygen therapy. On postoperative day 15, the tracheostomy tube was successfully removed. Cardiovascular support was fully withdrawn by postoperative day 10.

On postoperative day 18, the patient was transferred to the cardiac surgery department. A follow-up transthoracic echocardiography was performed on day 28, with findings summarized in Table. Endomyocardial biopsies were conducted on postoperative days 14 and 29. Both specimens showed no evidence of acute cellular rejection – graded as 0 according to the ISHLT classification – with no perivascular or interstitial infiltrates identified.

On postoperative day 33 (August 1, 2023), following comprehensive follow-up assessments, the patient was discharged from the hospital in stable condition.

DISCUSSION

Early graft dysfunction is a serious post-HT complication. According to the literature [9], three major categories of risk factors contribute to the development of primary dysfunction: donor-related factors, recipient-related factors, and perioperative/surgical factors.

In the present case, risk factors from all three categories were present. The recipient had a significant comorbidity – type 2 diabetes mellitus. From the donor side, moderate hypernatremia (serum sodium of 149 mmol/L) was documented. In addition, there was a gender mismatch between donor and recipient.

The combination of these risk factors likely triggered the development of biventricular graft dysfunction (BGD) in this patient.

The diagnosis of biventricular graft dysfunction in this case is supported by several clinical findings consistent with the consensus definition provided by the

Table

Comparative echocardiographic parameters of the transplanted heart

Parameters	Pre-transplant value	Post-transplant value	At discharge
LVEDV	4.9 cm	4.1 cm	5.4 cm
LVESV	3.3 cm	2.6 cm	3.6 cm
EF	63%	66%	56%
CF	34%	36%	33%
IVS thickness	1.1 cm, with basal segment thickening up to 1.3 cm	1.2 cm, with basal segment thickening up to 1.3 cm	1.3 cm
LVPWT	1.2 cm	1.1 cm	1.25 cm
Right ventricular systolic pressure	30–35 mmHg	35 mmHg	34 mmHg

Abbreviations: LVEDV, left ventricular end-diastolic diameter; LVESV, left ventricular end-systolic diameter; EF, ejection fraction; CF, contractility fraction; IVS, interventricular septal; LVPWT, left ventricular posterior wall thickness; RVSP, right ventricular systolic pressure.

International Society for Heart and Lung Transplantation (ISHLT) [10]:
- High levels of cardiotonic and vasopressor support, reflected by a VIS of 55 points.
- Persistent hemodynamic instability, evidenced by sustained hypotension; despite maximal inotropic therapy, arterial blood pressure remained no higher than 80/50 mmHg.
- Central hemodynamic parameters indicating biventricular dysfunction: right heart overload, demonstrated by elevated right ventricular and pulmonary artery pressures; and left ventricular dysfunction, reflected by PCWP at the upper limit of normal, coupled with low cardiac output and cardiac index.

Thus, the development of severe biventricular graft dysfunction influenced the clinical course of this case. The severe form of this condition is associated with high perioperative mortality. According to various studies [10, 11], the risk of death or the need for retransplantation varies with the severity of the dysfunction. In severe cases, the probability of death or retransplantation – often necessitating MCS in the early post-transplant period – ranges from 40% to 50%.

Regardless of the underlying cause or specific type of early graft dysfunction, the first-line treatment is pharmacological hemodynamic support. This involves the administration of inotropic and vasopressor agents to maintain adequate metabolic homeostasis, thereby providing a critical window for further diagnostic evaluation and intervention.

The VIS is a useful tool for objectively assessing the level of pharmacological circulatory support. The concept was originally introduced by Wernovsky et al. in 1995 [12], who developed an inotropic index based solely on inotropic agents. In 2010, Gaies et al. updated this scoring system by incorporating vasopressors, resulting in the modern VIS formula now widely used to evaluate the severity of graft dysfunction [13]. A more recent adaptation of the VIS, known as the Levosimendan VIS (LVIS), includes levosimendan as an additional parameter [14]. The formula is as follows:

LVIS = Dopamine (μg/kg/min) + Dobutamine (μg/kg/min) + 100 × Epinephrine (μg/kg/min) + 10 × Milrinone (μg/kg/min) + 10,000 × Vasopressin (units/kg/min) + 100 × Norepinephrine (μg/kg/min) + 50 × Levosimendan (μg/kg/min).

Failure to achieve hemodynamic stability with drug treatment suggests severe graft dysfunction, which is clinically equivalent to severe cardiogenic shock and may necessitate the initiation of MCS. A rising VIS can indicate worsening clinical status and treatment ineffectiveness. Although VIS alone should not serve as the sole criterion for initiating MCS [15], a score ≥32 points has been associated with delayed initiation of MCS and increased mortality risk [16].

MCS serves several critical functions in the management of severe graft dysfunction:
- Enhancing systemic perfusion;
- Improving coronary perfusion;
- Reducing left ventricular filling pressure (decreasing wall tension and myocardial oxygen demand).

Several retrospective studies have demonstrated the advantages of ECMO over other circulatory support modalities in such clinical scenarios [17, 18].

Acute kidney injury (AKI) is another serious complication that we encountered in this case. Among the established risk factors for AKI [19], the most relevant in this patient were prolonged CPB time, pre-existing diabetes mellitus, and the use of ECMO.

The potential for ECMO-related AKI was a key consideration in the decision to wean the patient early from MCS. On one hand, discontinuing ECMO helped eliminate the extracorporeal circuit as a contributing factor to kidney injury. On the other hand, MCS plays a vital role in improving renal perfusion by supporting cardiac output and ensuring adequate oxygenation, which together contribute to improved tissue oxygen delivery to the tissues.

AKI is a relatively common complication in heart transplant recipients, with an incidence reported as high as 47.1% [20]. When AKI develops and necessitates treatment, the choice of an appropriate therapeutic approach becomes critical. According to the 2012 KDIGO guidelines for AKI in critically ill patients [21], prolonged renal replacement therapy (RRT) modalities are preferred in cases of hemodynamic instability. In this patient, prolonged veno-venous hemodiafiltration was implemented in accordance with these guidelines. This preference is based on the slower rate of fluid removal and the absence of fluid migration, which occurs with the rapid removal of dissolved substances.

The simultaneous use of MCS and prolonged RRT has been previously discussed by Ostermann et al. [22], who noted that current evidence does not conclusively demonstrate a reduction in mortality with this combination.

However, in contrast to these findings, we observed an improvement in hemodynamic stability after the use of RRT in our patient. This was evidenced by a marked reduction in the need for cardiotonic support.

The timing of RRT initiation remains a subject of ongoing debate. However, Poz et al. [23] argue that early initiation may offer the greatest benefit, particularly by maximizing the therapeutic potential of the method.

In the present case, cardiorenal syndrome played a pivotal role in the clinical trajectory [24]. This syndrome arises from the bidirectional relationship between cardiac and renal dysfunction, whereby acute or chronic failure of one organ precipitates or exacerbates dysfunction in the other. In this patient, severe graft dysfunction of the transplanted heart precipitated renal failure. Given this

dynamic, a multimodal treatment approach, incorporating both MCS and prolonged RRT, was likely instrumental in achieving the favorable clinical outcome observed in this case.

CONCLUSIONS

The combined use of MCS – which ensures adequate oxygen delivery to tissues – and prolonged RRT – which facilitates the removal of metabolic waste, inflammatory mediators, and hemolytic byproducts – may contribute to improved clinical outcomes and supports the recovery of vital organ function.

This simultaneous application of MCS and RRT may improve clinical outcomes, which requires further research.

Early initiation of RRT in patients with transplant cardiac dysfunction requiring MCS shows promise and warrants further investigation.

Based on the findings from the presented clinical case, it is essential to adopt an individualized and multidisciplinary approach to the intensive care management of acute heart failure secondary to heart transplant dysfunction.

The authors declare no conflict of interest.

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